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EXAMINER	
BLANCHARD, DAVID J	
ART UNIT	**PAPER NUMBER**
1642	

DATE MAILED: 09/23/2004

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	09/230,111	SATO ET AL.	
	Examiner	Art Unit	
	David J Blanchard	1642	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If the period for reply specified above is less than thirty (30) days, a reply within the statutory minimum of thirty (30) days will be considered timely.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *07 June 2004*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *121-132,140 and 141* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *121-132, 140-141* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

1. Claims 121 and 140 have been amended.

2. Claims 121-132 and 140-141 are pending and under examination.

The claims are being examined to the extent that they are drawn to a method of identifying a compound that inhibits specific binding between a signal-transducing protein and a cytoplasmic protein or a method of identifying a compound that displaces a cytoplasmic protein bound to a signal-transducing protein or displacing a signal-transdusing protein bound to a cytoplasmic protein. If Applicant wishes to claim the composition comprising the peptides of SEQ ID NOS:9 and 11-16 identified by the claimed method as inhibiting the specific interaction between Fas and FAP1, Applicant should file a continuation application. See new matter rejection below (item #12).

3. The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

4. This Office Action contains New Grounds of Rejections.

Objections/Rejections Withdrawn

5. The objection to the specification for containing sequence disclosures not in compliance with the requirements of 37 C.F.R. 1.821-1.825 is withdrawn in view of the amendments to the specification and the sequence listing filed 6/7/2004.

6. The rejection of claims 121-132 and 140-141 under 35 U.S.C. 112, first paragraph, because the specification while being enabling for a method of identifying compounds that disrupt the binding of Fas with FAP, does not reasonably provide

enablement for methods of screening for compounds that disrupt the binding of a CD4 receptor, a P75 receptor, a serotonin receptor, a serotonin 2B receptor, a NMDA receptor, or a K+ channel or a composition comprising a peptide selected from SEQ ID NOS:9 and 11-16 is withdrawn in view of Applicant's arguments.

7. The rejection of claims 121-132 and 140-141 (item # 13 of the previous Office Action, mailed 3/5/04) under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention is withdrawn in view of the amendments to the claims.

8. The rejection of claim 140 (item #16 of the previous Office Action, mailed 3/5/04) under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention is withdrawn in view of the amendments to the claim.

9. The rejection of claims 121-132 and 140-141 (part a) under 35 U.S.C. 112, first paragraph, NEW MATTER, as containing subject matter, which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventors, at the time the application was filed, had possession of the claimed invention is withdrawn in view of the amendments to the claims.

10. The rejection of claims 121-132 and 140-141 under 35 U.S.C. 112, first paragraph, written description (item #19 of the previous Office Action, mailed 3/5/2004), as containing subject matter, which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventors, at the time

the application was filed, had possession of the claimed invention is withdrawn in view of Applicant's arguments.

Response to Arguments

11. The rejection of claims 121-132 and 140-141 under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention is maintained.

a. With respect to the indefiniteness of the phrase "and the signal-transducing protein bound to the cytoplasmic protein" (see item #14 of the Office Action, mailed 3/5/2004), the response filed 6/7/2004 has been carefully considered, but is deemed not to be persuasive. The response argues that the claims have been amended to more clearly define the claimed methods, thereby obviating the rejection. In response to this argument claim 121 remains completely ambiguous and indefinite. As an initial matter, claim 121 is improperly drafted not clear because it encompasses two entirely different methods in the same claim. First, the preamble of claim 121 recites a method of identifying a compound that inhibits specific binding between a signal-transducing protein and a cytoplasmic protein. The claim then goes on (see part (a)) to recite that "contacting the cytoplasmic protein bound to the signal-transducing protein with a plurality of compounds...". Thus, the preamble of claim 121 is directed towards a method of identifying a compound that inhibits the formation of a complex comprising a cytoplasmic protein and a signal-transducing protein (i.e., inhibits the specific binding), whereas part (a) of claim 121 encompasses disrupting the cytoplasmic protein already

bound to the signal-transducing protein by contacting the cytoplasmic protei-signal-transducing protein complex with a compound (i.e., displaces/dissociates the signal-transducing protein bound to the cytoplasmic protein). Therefore, part (a) of claim 121 falls outside the scope encopmpassed by the preamble of claim 121, which is "A method of identifying a compound that inhibits specific binding between a signal-transducing protein and a cytoplasmic protein" [i.e., a compound that inhibits the formation of a cytoplasmic protein-signal-transducing protein complex] and not a method of displacing a cytoplasmic protein already bound to a signal-transducing protein with a compound known to displace the signal-transducing protein bound to the cytoplasmic protein. Again, the claims are totally unclear because as detailed above, part (a) of claim 121 is not consistent and departs from the method contemplated by the preamble of the claim. As written, claim 121 is drawn to two disparate methods, one method is for identifying a compound having a specific function, the second method is for using a known compound known to disrupt a specific protein-protein complex (i.e., cytoplasmic protein-signal-transducing protein complex). Claims 123 and 124 are similar to claim 121 as explained in detail above because they recite "the specific binding between the signal-transducing protein and the cytoplasmic protein is inhibited and the cytoplasmic protein is displaced." Again, as above, if the specific binding between the signal-transducing protein and the cytoplasmic protein is inhibited, the complex between the signal-transducing protein and the cytoplasmic protein is not formed. Accordingly, if the complex between the signal-transducing protein and the cytoplasmic protein is not formed, the cytoplasmic protein cannot be

displaced/dissociated. In order to displace a component of an interaction, that interaction must already exist. It is noted that claim 122 is consistent with the preamble of claim 121 (i.e., method of inhibiting the interaction between a cytoplasmic protein and a signal-transducing protein).

b. With respect to the indefinite nature of the phrase "a method of identifying a compound" and for reciting "known compound" in claim 121 (item #15 of the previous Office Action, mailed 3/5/2004), the response filed 6/7/2004 has been carefully considered, but is deemed not to be persuasive. The response argues that the claims have been amended to more clearly define the claimed methods, thereby obviating the rejection. In response to this argument the amendments to claim 121 changed the phrase "a known compound previously shown to be able to" to recite "a compound known to be able to", which is merely a different way of saying the same thing and therefore does not obviate the instant rejection. Claim 121 is unclear because the preamble of claim 121 is drawn to "A method of identifying a compound that inhibits specific binding between a signal-transducing protein and a cytoplasmic protein...", meaning that the method is directed toward identifying some unknown compound that is able to inhibit the interaction between the two proteins. Thus, part (a) is unclear because it recites using a compound known to displace the signal-transducing protein bound to the cytoplasmic protein or displace/dissociate cytoplasmic protein bound to the signal-transducing protein. Why is the method of identifying a compound (i.e., an unknown compound) using a compound known to displace/dissociate the signal-

transducing protein bound to the cytoplasmic protein or displace cytoplasmic protein bound to the signal-transducing protein? As written, the metes and bounds of the claims cannot be determined.

It is noted that the specification does not apparently teach any compound known to displace the cytoplasmic protein bound to the signal-trasnducing protein or displace a signal-transducing protein bound to a cytoplasmic protein. The specification only teaches a method of identifying a compound (i.e., peptides from a random peptide library) that inhibits the specific binding between Fas and FAP-1 (see Figures 2-3 and pages 24-32). Applicant is invited to specifically point out in the instant specification where it is shown that a known compound displaces a cytoplasmic protein bound to a signal-transducing protein or displaces a signal-transducing protein bound to a cytoplasmic protein as recited in part (a) of claim 121.

12. The rejection of claims 121-132 and 140-141 under 35 U.S.C. 112, first paragraph, NEW MATTER (part b), as containing subject matter, which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventors, at the time the application was filed, had possession of the claimed invention is maintained.

The response filed 6/7/2004 has been carefully considered, but is deemed not to be persuasive. The response argues and directs the examiner to page 6, lines 9-28, page 11, lines 26-31, and Figure 2 where SEQ ID NOS:9 and 11-16 are disclosed specifically as interacting with the PDZ domain of FAP1. In response to this argument

the pages cited by applicant disclose a "composition" comprising the recited peptide sequences, however, the claims are drawn to a method of identifying a compound that inhibits specific binding between a signal-transducing protein and a cytoplasmic protein. If Applicant wishes to claim the composition comprising the peptides of SEQ ID NOS:9 and 11-16 identified by the claimed method as inhibiting the specific interaction between Fas and FAP1, applicant should file a continuation application. There is no support in the specification or claims as originally filed for the peptides of SEQ ID NOS:9 and 11-16 in a method of identifying a compound that inhibits specific binding between a signal-transducing protein and a cytoplasmic other than FAP1 and Fas, respectively. The only interaction that the peptides of SEQ ID NOS:9 and 11-16 were shown to inhibit is the FAP1-Fas interaction (see Figure 2).

New Grounds of Rejections

13. Claims 140 and 141 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method of identifying a compound that inhibits specific binding between a signal-transducing protein and a cytoplasmic protein or a method of dissociating a cytoplasmic protein bound to a signal-transducing protein, wherein the signal-transducing protein is a CD4 receptor, a p75 receptor, a serotonin 2A receptor, a serotonin 2B receptor, a NMDA receptor or a K+ channel and the cytoplasmic protein is the respective ligand comprising the amino acid sequence (G/S/A/E)-L-G-(F/I/L) (SEQ ID NO:1) or wherein the cytoplasmic protein-signal-transducing protein complex is Fas-FAP-1 or p75-FAP-1, does not reasonably provide

enablement for a method of identifying a compound that inhibits specific binding between a signal-transducing protein and a cytoplasmic protein and a method of dissociating a cytoplasmic protein bound to a signal-transducing protein, wherein the cytoplasmic protein is FAP-1 and the signal-transducing protein is a CD4 receptor, a serotonin 2A receptor, a serotonin 2B receptor, a NMDA receptor or a K+ channel. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

Factors to be considered in determining whether undue experimentation is required, are summarized in Ex parte Forman, 230 USPQ 546 (BPAI 1986). They include the nature of the invention, the state of the prior art, the relative skill of those in the art, the amount of direction or guidance disclosed in the specification, the presence or absence of working examples, the predictability or unpredictability of the art, the breadth of the claims, and the quantity of experimentation which would be required in order to practice the invention as claimed.

The claims are broadly drawn to a method of identifying a compound that inhibits specific binding between a signal-transducing protein and a cytoplasmic protein or a method of dissociating a cytoplasmic protein bound to a signal-transducing protein, wherein the signal-transducing protein is a CD4 receptor, a p75 receptor, a serotonin 2A receptor, a serotonin 2B receptor, a NMDA receptor or a K+ channel and the cytoplasmic protein is the respective ligand comprising the amino acid sequence (G/S/A/E)-L-G-(F/I/L) (SEQ ID NO:1) or wherein the cytoplasmic protein is FAP-1.

The specification teaches a method of identifying a compound that inhibits specific binding between Fas (signal-transducing protein) and FAP-1 (cytoplasmic protein) and FAP-1 contains six GLGF (PDZ/DHR) repeats, wherein the third GLGF motif shows a specific interaction with the C-terminus of the Fas receptor (see Figure 2 and pages 24-32 and page 2, lines 30-35). The specification teaches that FAP-1 also interacts with the nerve growth factor receptor (p75) (see Figures 10-12). The specification also teaches a limited number of cytoplasmic proteins and their respective signal-transducing protein interaction partners (see Table 1).

The specification does not teach a method of identifying a compound that inhibits specific binding between a signal-transducing protein and a cytoplasmic protein other than the Fas-FAP-1 interaction. The specification does not teach any method of displacing/dissociating a cytoplasmic protein bound to a signal-transducing protein or displacing/dissociating a signal-transducing protein bound to a cytoplasmic protein. The specification does not teach the interaction of FAP-1 with a CD4 receptor, a serotonin 2A receptor, a serotonin 2B receptor, a NMDA receptor or a K+ channel. There are no working examples in applicants specification to guide the skilled artisan in practicing a method of identifying a compound that inhibits the interaction between FAP-1 and a CD4 receptor, a serotonin 2A receptor, a serotonin 2B receptor, a NMDA receptor or a K+ channel or a method of dissociating a FAP-1 bound to a CD4 receptor, a p75 receptor, a serotonin 2A receptor, a serotonin 2B receptor, a NMDA receptor or a K+ channel. The specification does not enable a method of identifying a compound that inhibits the interaction between FAP-1 and a CD4 receptor, a serotonin 2A receptor, a

serotonin 2B receptor, a NMDA receptor or a K+ channel or a method of dissociating a FAP-1 bound to a CD4 receptor, a serotonin 2A receptor, a serotonin 2B receptor, a NMDA receptor or a K+ channel.

The state of the prior art is such that PDZ motifs from different types of proteins (cytoplasmic proteins) can recognize unique carboxyl-terminal peptide motifs (signal-trasnducing proteins/receptors), the *in vivo* relevance of most of them remains to be elucidated (see page 30215, right column; Cuppen et al, The Journal of Biological Chemistry, 272(48):30215-30220, 1997). Accordinhg to Cuppen et al, the human and mouse Fas carboxyl-termini differ by two of the three C-terminal amino acids (see Figure 1) and have different PDZ reactivity (see page 30216, right column). Two nonrelated domains, the nNOS PDZ motif, found to recognize the carboxyl-terminal tG-(D/E)-X-V motif and the RIL PDZ motif, did not interact with mouse or human Fas receptor (Cuppen et al, see page 30216, right column). Another motif, the second PDZ motif of PSD95/SAP90, known to recognize t(T/S)-X-V did interact only with the human Fas receptor (see page 30216, right column). Thus, distinct PDZ motifs bind to distinct optimal sequences of signal-transducing proteins. Yanagisawa et al (The journal of Biological Chemistry, 272(13):8539-8545, 1997) teach that the third PDZ repeat of FAP-1 shows a specific interaction with the C terminus of Fas receptor and the third PDZ domain of FAP-1 has the amino acid sequence SLGI instead of GLGF (see page 8544). The prior art does not teach or fairly suggest any signal-transducing protein other than FAP-1, which contains the amino acid sequence SLGI.

The level of skill in the art is acknowledged to be high, however, due to the high degree of uncertainty in predicting which signal-transducing proteins interact with FAP-1, and the lack of teaching in the specification as to a method for identifying a compound that inhibits specific binding between FAP-1 and a CD4 receptor, a serotonin 2A receptor, a serotonin 2B receptor, a NMDA receptor or a K+ channel or a method of dissociating a FAP-1 bound to a CD4 receptor, a p75 receptor, a serotonin 2A receptor, a serotonin 2B receptor, a NMDA receptor, a K+ channel or Fas, it would require undue experimentation by one skilled in the art to practice a method of identifying a compound that inhibits the interaction between FAP-1 and a CD4 receptor, a serotonin 2A receptor, a serotonin 2B receptor, a NMDA receptor or a K+ channel or a method of dissociating a FAP-1 bound to a CD4 receptor, a serotonin 2A receptor, a serotonin 2B receptor, a NMDA receptor or a K+ channel.

Claim Rejections - 35 USC § 103

14. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

The factual inquiries set forth in *Graham* **v.** *John Deere Co.*, 383 U.S. 1, 148 USPQ 459 (1966), that are applied for establishing a background for determining obviousness under 35 U.S.C. 103(a) are summarized as follows:

1. Determining the scope and contents of the prior art.
2. Ascertaining the differences between the prior art and the claims at issue.
3. Resolving the level of ordinary skill in the pertinent art.
4. Considering objective evidence present in the application indicating obviousness or nonobviousness.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

15. Claims 121-132 are rejected under 35 U.S.C. 103(a) as being unpatentable over Reed et al (U.S. Patent 5,876,939, priority at least to 3/27/1995 cited on previous PTO-892 mailed 10/31/2001) as evidenced by Niethammer et al (The Journal of Neuroscience, 16(7):2157-2163, 1996) in view of Kornau et al (Science, 269(5231):1737-1740, 22 September 1995).

Due to the indefinite nature of the claims, the claims are interpreted as being drawn to a method of identifying a compound that inhibits specific binding between a signal-transducing protein, which is a CD4 receptor, a p75 receptor, a serotonin 2A receptor, a serotonin 2B receptor, a NMDA receptor or a K+ channel and their respective cytoplasmic protein containing the amino acid sequence (G/S/A/E)-L-G-(F/I/L) (SEQ ID NO:1), wherein the inhibition affects the transcription activity of a

reporter gene and wherein the cytoplasmic protein or the compound is bound to a solid support and the compound comprises an antibody, an inorganic compound, an organic compound, a peptide, a peptidomimetic, a polypeptide or a protein. Further, the claimed method can be performed in vitro or in vivo, in a yeast cell or a mammalian cell.

The claims are also interpreted as being drawn to a method of dissociating a bound cytoplasmic protein-signal-transducing protein complex, wherein the cytoplasmic protein comprises the amino acid sequence (G/S/A/E)-L-G-(F/I/L) (SEQ ID NO:1) and the signal-transducing protein is a CD4 receptor, a p75 receptor, a serotonin 2A receptor, a serotonin 2B receptor, a NMDA receptor or a K+ channel, wherein the inhibition affects the transcription activity of a reporter gene and wherein the cytoplasmic protein or the compound is bound to a solid support and the compound comprises an antibody, an inorganic compound, an organic compound, a peptide, a peptidomimetic, a polypeptide or a protein. Further, the claimed method can be performed in vitro or in vivo, in a yeast cell or a mammalian cell.

Reed et al teach a method of identifying an agent (i.e., a compound) that interferes with the ability of a FAP (Fas-associated proteins, which are alternatively spliced forms of a protein tyrosine phosphatase; see column 6, lines 30-34) to interact with a Fas or the agent (i.e., compound) can act by causing the dissociation of a bound FAP-Fas complex and the agent or compound can be an organic molecule, a peptide, a peptido-mimetic, or a protein (see column 13, line 51-column 14, line 20). The screening assay can be performed in vivo using the yeast two-hybrid system, wherein an effective agent (i.e., compound) alters the level of transcription of a reporter gene or

the assay can be performed in vitro and the assay can be adapted for use in mammalian cells (see column 15 and claims). Reed et al teach that the FAP proteins comprise multiple GLGF domains (see Figure 7), which are also known as PDZ domains as evidenced by Niethammer et al (see abstract). Reed et al also teach that the in vitro assay for identifying an agent that alters the association of FAP and Fas wherein one of the components is bound to a solid support (see column 15, line 48-column 16, line 21). Reed et al do not specifically teach the signal-transducing proteins such as a CD4 receptor, a p75 receptor, a serotonin receptor, a NMDA receptor or a K+ channel that interact with cytoplasmic proteins containing PDZ domains. This deficiency is made up for in the teachings of Kornau et al.

Kornau et al teach signal-transducing receptors including Fas, p75, serotonin, NMDA and K+ channels that have a carboxyl-terminal tSXV motif, which interfaces or binds with PDZ domains and Kornau et al proposes that tSXV-PDZ domain interactions play a general role in connecting receptors and channels to signal-transduction machineries (see entire document, particularly Table 1).

It would have been prima facie obvious to one of ordinary skill in the art at the time the claimed invention was made to use the signal-transduction proteins as taught by Kornau et al in a method of identifying a compound that interferes with the ability of a signal-transducing protein to specifically bind its respective cytoplasmic protein or a method of disrupting/dissociating a signal-transduction protein-cytoplasmic protein complex as taught by Reed et al, wherein the cytoplasmic protein comprises a GLGF domain, also known as a PDZ domain as evidenced by Niethammer et al.

One of ordinary skill in the art would have been motivated to and had a reasonable expectation of success to use the signal-transduction proteins as taught by Kornau et al in a method of identifying a compound that interferes with the ability of a signal-transducing protein to specifically bind its respective cytoplasmic protein or a method of disrupting/dissociating a signal-transduction protein-cytoplasmic protein complex as taught by Reed et al, wherein the cytoplasmic protein comprises a GLGF domain, also known as a PDZ domain as evidenced by Niethammer et al because Reed et al teach a method of identifying an agent/compound that interferes with the ability of FAP (i.e., a cytoplasmic protein), which comprises the amino acid sequence GLGF (also known as a PDZ domain as evidenced by Niethammer et al; see abstract) to specifically bind Fas (i.e., a signal-transducing protein) or the agent (i.e., compound) can act by causing the dissociation of a bound FAP-Fas complex and Kornau et al teach that Fas as well as other signal-transducing receptors including p75, serotonin, NMDA and K+ channels have a carboxyl-terminal tSXV motif that interfaces or binds with PDZ domains, also known as GLGF domains as evidenced by Niethammer et al. Thus, it would have been obvious to one skilled in the art at the time the invention was made to use the signal-transduction proteins as taught by Kornau et al in a method of identifying a compound that interferes with the ability of a signal-transducing protein to specifically bind its respective cytoplasmic protein or a method of disrupting/dissociating a signal-transduction protein-cytoplasmic protein complex as taught by Reed et al, wherein the cytoplasmic protein comprises a GLGF domain, also known as a PDZ domain.

Therefore, the invention as a whole was prima facie obvious to one of ordinary skill in the art at the time the invention was made, as evidenced by the references.

Conclusions

16. No claim is allowed.

17. Any inquiry concerning this communication or earlier communications from the examiner should be directed to David J. Blanchard whose telephone number is (571) 272-0827. The examiner can normally be reached at Monday through Friday from 8:00 AM to 6:00 PM, with alternate Fridays off. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Jeffrey Siew, can be reached at (571) 272-0787. The official fax number for the organization where this application or proceeding is assigned is 703-872-9306.

Information regarding the status of an application may be obtained from the patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Respectfully,
David J. Blanchard
571-272-0827

LARRY R. HELMS, PH.D
PRIMARY EXAMINER